an assessment of the possible association of heart disease and passive smoking. EPA is considering whether such an assessment should be undertaken in the future, but has no plans to do so at this time.

Top of page

Scientific Approach

EPA reached its conclusions concerning the potential for ETS to act as a human carcinogen based on an analysis of all of the available data, including more than 30 epidemiologic (human) studies looking specifically at passive smoking as well as information on active or direct smoking. In addition, EPA considered animal data, biological measurements of human uptake of tobacco smoke components and other available data. The conclusions were based on what is commonly known as the total weight-of-evidence" rather than on any one study or type of study.

The finding that ETS should be classified as a Group A carcinogen is based on the conclusive evidence of the dose-related lung carcinogenicity of mainstream smoke in active smokers and the similarities of mainstream and sidestream smoke given off by the burning end of the cigarette. The finding is bolstered by the statistically significant exposure-related increase in lung cancer in nonsmoking spouses of smokers which is found in an analysis of more than 30 epidemiology studies that examined the association between secondhand smoke and lung cancer.

The weight-of-evidence analysis for the non-cancer respiratory effects in children is based primarily on a review of more than 100 studies, including 50 recent epidemiology studies of children whose parents smoke.

Top of page

Beyond the Risk Assessment

Although EPA does not have any regulatory authority for controlling ETS, the Agency expects this report to be of value to other health professionals and policymakers in taking appropriate steps to minimize peoples' exposure to tobacco smoke in indoor environments.

In cooperation with other government agencies, EPA will continue its education and outreach program to inform the public and policy makers on what to do to reduce the health risks of ETS as well as other indoor air pollutants.

Top of page

For Further Information

A limited number of copies of the complete report can be obtained free of charge from:

Center for Environmental Research Information (CERI)
U.S. EPA
26 W. Martin Luther King Drive
Cincinnati, OH 45268
Telephone: 513-569-7562
Fax: 513-569-7566
Ordering Number: EPA/600/6-90/006F

The report Respiratory Health Effects of Passive Smoking: Lung Cancer and Other Disorders is available via a series of Adobe Acrobat PDF files from EPA's Office of Research and Development (ORD)

You can also get a copy of the report from:

Indoor Air Quality Information Clearinghouse (IAQ INFO)
P.O. Box 37133,
Washington D.C. 20013-7133
Telephone: 1-800-438-4318 or (703) 356-4020
Fax: (703) 356-5386 or
e-mail: iaqinfo@aol.com

A number of government agencies can provide additional information addressing the health risks of environmental tobacco smoke. These include:

Office on Smoking and Health/Centers for Disease Control EXIT disclaimer
Center for Chronic Disease Prevention and Health Promotion
Mail Stop K-50, 4770 Buford Highway
Atlanta, GA 30341
1-800-CDC-1311

National Cancer Institute EXIT disclaimer
Building 31, Room 10A24
Bethesda, MD 20892
1-800-4-CANCER

The National Heart, Lung, and Blood Institute EXIT disclaimer
Information Center
4733 Bethesda Avenue, Suite 530
Bethesda, MD 20814

National Institute for Occupational Safety and Health EXIT disclaimer
4676 Columbia Parkway
Cincinnati, Ohio 45226-1998
1-800-35-NIOSH

Top of page

Smoke-free Homes
Where You Live
A to Z Subject Index
Smoke-free Homes
Health Effects
The Smoke-free Homes Pledge
Prometa no fumar en su hogar
Developing Smoke-free Homes Programs
Frequent Questions
Publications and Materials
Links
Contact Us

"Setting the Record Straight: Secondhand Smoke is a Preventable Health Risk"

Office of Air and Radiation
Office of Radiation and Indoor Air
Indoor Environments Division (6609J)
EPA Document Number 402-F-94-005, June 1994

Contents

Background/Statistics/Conclusions

In early 1993, EPA released a report (Respiratory Health Effects of Passive Smoking: Lung Cancer and Other Disorders; EPA/600/6-90/006 F) that evaluated the respiratory health effects from breathing secondhand smoke (also called environmental tobacco smoke (ETS)). In that report, EPA concluded that secondhand smoke causes lung cancer in adult nonsmokers and impairs the respiratory health of children. These findings are very similar to ones made previously by the National Academy of Sciences and the U.S. Surgeon General.

The EPA report classified secondhand smoke as a Group A carcinogen, a designation which means that there is sufficient evidence that the substance causes cancer in humans. The Group A designation has been used by EPA for only 15 other pollutants, including asbestos, radon, and benzene. Only secondhand smoke has actually been shown in studies to cause cancer at typical environmental levels. EPA estimates that approximately 3,000

American nonsmokers die each year from lung cancer caused by secondhand smoke.

Every year, an estimated 150,000 to 300,000 children under 18 months of age get pneumonia or bronchitis from breathing secondhand tobacco smoke. Secondhand smoke is a risk factor for the development of asthma in children and worsens the condition of up to one million asthmatic children.

EPA has clear authority to inform the public about indoor air pollution health risks and what can be done to reduce those risks. EPA has a particular responsibility to do everything possible to warn of risks to the health of children.

A recent high profile advertising and public relations campaign by the tobacco industry may confuse the American public about the risks of secondhand smoke. EPA believes it's time to set the record straight about an indisputable fact: secondhand smoke is a real and preventable health risk.

EPA absolutely stands by its scientific and well documented report. The report was the subject of an extensive open review both by the public and by EPA's Science Advisory Board (SAB), a panel of independent scientific experts. Virtually every one of the arguments about lung cancer advanced by the tobacco industry and its consultants was addressed by the SAB. The panel concurred in the methodology and unanimously endorsed the conclusions of the final report.

The report has also been endorsed by the U.S. Department of Health and Human Services, the National Cancer Institute, the Surgeon General, and many major health organizations.

Top of page

Classification of Secondhand Smoke as a Known Human (Group A) Carcinogen

The finding that secondhand smoke causes lung cancer in nonsmoking adults is based on the total weight of the available evidence and is not dependent on any single analysis. This evidence includes several important facts.

First, it is indisputable that smoking tobacco causes lung cancer in humans, and there is no evidence that there is a threshold below which smoking will not cause cancer.

Second, although secondhand smoke is a dilute mixture of mainstream" smoke exhaled by smokers and sidestream" smoke from the burning end of a cigarette or other tobacco product, it is chemically similar to the smoke inhaled by smokers, and contains a number of carcinogenic compounds.

Third, there is considerable evidence that large numbers of people who do not smoke are exposed to, absorb, and metabolize significant amounts of secondhand smoke.

Fourth, there is supporting evidence from laboratory studies of the ability of secondhand smoke both to cause cancer in animals and to damage DNA, which is recognized by scientists as being an instrumental mechanism in cancer development.

Finally, EPA conducted multiple analyses on the then-available 30 epidemiology studies from eight different countries which examined the association between secondhand smoke and lung cancer in women who never smoked themselves but were exposed to their husband's smoke. Since the epidemiology studies are the major thrust of the tobacco industry arguments against the EPA report, these studies are examined in more detail below.

Top of page

The Epidemiology Studies

The most important aspect of the review of the epidemiology studies is the remarkable consistency of results across studies that support a causal association between secondhand smoke and lung cancer.

In assessing the studies several different ways, it becomes clear that the extent of the consistency defies attribution to chance. When looking only at the simple measure of exposure of whether the husband ever smoked, 24 of 30 studies reported an increase in risk for nonsmoking women with smoking husbands. Since many of these studies were small, the chance of declaring these increases statistically significant was small. Still, nine of these were statistically significant, and the probability that this many of the studies would be statistically significant merely by chance is less than *1 in 10 thousand*.

The simple overall comparison of risks in ever vs. never exposed to spousal smoking tends to hide true increases in risk in two ways. First, it categorizes many women as never exposed who actually received exposure from sources other than spousal smoking. It also includes some women as exposed who actually received little exposure from their husband's smoking. One way to correct for this latter case is to look at the women whose husbands smoked the most. When one looks at the 17 studies that examined cancer effects based on the level of exposure of the subjects, every study found an increased lung cancer risk among those subjects who were most exposed. Nine were statistically significant. The probability of 9 out of 17 studies showing statistically significant results occurring by chance is less than *1 in ten million.*

Probably the most important finding for a causal relationship is one of increasing response with increasing exposure, since such

associations cannot usually be explained by other factors. Such exposure-response trends were seen in all 14 studies that examined the relationship between level of exposure and effect. In 10 of the studies the trends were statistically significant. The probability of this happening by chance is less than *1 in a billion*.

It is unprecedented for such a consistency of results to be seen in epidemiology studies of cancer from environmental levels of a pollutant. One reason is that it is extremely difficult to detect an effect when virtually everyone is exposed, as is the case with secondhand smoke. However, consistent increased risks for those most exposed and consistent trends of increasing exposure showing an increasing effect provide strong evidence that secondhand smoke increases the risk of lung cancer in nonsmokers.

Top of page

How Big a Lung Cancer Risk for Adults?

The evidence is clear and consistent: secondhand smoke is a cause of lung cancer in adults who don't smoke. EPA has never claimed that minimal exposure to secondhand smoke poses a huge individual cancer risk. Even though the lung cancer risk from secondhand smoke is relatively small compared to the risk from direct smoking, unlike a smoker who chooses to smoke, the nonsmoker's risk is often involuntary. In addition, exposure to secondhand smoke varies tremendously among exposed individuals. For those who must live or work in close proximity to one or more smokers, the risk would certainly be greater than for those less exposed.

EPA estimates that secondhand smoke is responsible for about 3,000 lung cancer deaths each year among nonsmokers in the U.S.; of these, the estimate is 800 from exposure to secondhand smoke at home and 2,200 from exposure in work or social situations.

Top of page

The Risks to Children are Widely Acknowledged

The conclusion that secondhand smoke causes respiratory effects in children is widely shared and virtually undisputed. Even the tobacco industry does not contest these effects in its media and public relations campaign.

EPA estimates that every year, between 150,000 and 300,000 children under 1-1/2 years of age get bronchitis or pneumonia from breathing secondhand tobacco smoke, resulting in thousands of hospitalizations. In children under 18 years of age, secondhand smoke exposure also results in more coughing and wheezing, a small but significant decrease in lung function, and an increase in fluid in the middle ear. Children with asthma have more frequent

and more severe asthma attacks because of exposure to secondhand smoke, which is also a risk factor for the onset of asthma in children who did not previously have symptoms.

Top of page

Other Risks

Secondhand smoke contains strong irritants and sensitizers and many adults, as well as children, suffer irritation and other acute effects whenever they are exposed to secondhand smoke. In addition, there is mounting evidence that exposure to secondhand smoke can have an effect on the cardiovascular system, although the EPA report does not address this issue.

Top of page

Tobacco Industry Media Campaign

The tobacco industry is raising numerous issues which may distract the public from the fact that secondhand smoke poses a real and preventable health risk. The tobacco industry neither acknowledges nor disputes EPA's conclusions of respiratory effects in children. It focuses instead on EPA's findings on lung cancer.

The overall thrusts of the tobacco industry's arguments are that EPA manipulated the lung cancer data to come to a predetermined conclusion. The industry also argues that a nonsmoker's exposure to secondhand smoke is so small as to be insignificant. The argument on minimal exposure is belied both by the acute irritation and respiratory effects and the fallacy of the cigarette equivalents" approach discussed below. Responses to the specific criticisms of EPA's assessment of the lung cancer data follow.

Top of page

The 11 U.S. Lung Cancer Studies

Critics of the EPA report argue that by normal statistical standards, none of the 11 U.S. studies included in the EPA report showed a statistically significant increase in the simple overall risk measure, and that EPA should therefore have been unable to conclude that secondhand smoke causes lung cancer in nonsmokers. These critics are misrepresenting a small part of the total evidence on secondhand smoke and lung cancer.

The consistency of study results in the highest exposure category and exposure-response trends discussed above also apply to the U.S. studies. For example, seven of the 11 U.S. studies had fewer than 45 cases, making statistical comparisons difficult. Nonetheless,

eight of the 11 had increased overall risks, and for the seven studies which reported on risks by amount of exposure, the highest exposure groups in all seven had increased risks. While the 11 U.S. studies are not, by themselves, conclusive, they do support the conclusion that secondhand smoke is causally associated with lung cancer.

Top of page

Studies Completed Since Release of the EPA Report

Critics claim that had EPA not excluded" the recent Brownson study, the Agency could not have concluded that secondhand smoke causes cancer. In fact, four new lung cancer epidemiology studies, including the Brownson study, have been published since the literature review cutoff date for the 1993 EPA report, and all support EPA's conclusions. Three of these are large U.S. studies funded, at least in part, by the National Cancer Institute. A 1992 study of Florida women by Stockwell et al. found a 60% overall increased risk of lung cancer from exposure to their husband's smoke, with significant results for both the highest exposure group and the exposure-response trend. The 1992 study of Missouri women by Brownson et al. found no overall increased risk, but did demonstrate a significant increase in risk in the highest spousal smoking exposure group and a positive exposure-response trend.

The 1994 study by Fontham et al. of women in two California and three Southern cities is the largest case-control study on the subject ever conducted and is considered by EPA to be the best designed study on secondhand smoke and lung cancer conducted to date. This study found significantly increased risks for overall exposure and in the highest exposure group and a strong positive exposure-response relationship. These findings were significant not only for exposure from spouses, but also for exposure in the workplace and in social situations.

Top of page

90% vs. 95% Confidence Intervals

Critics of the EPA report have charged that EPA changed the confidence interval in order to come to a predetermined conclusion. However, the conclusion that secondhand smoke is a known human carcinogen simply does not hinge on whether or not a 95% or 90% confidence interval" was used. A confidence interval is used to display variability in relative risk estimates in the epidemiology studies. As discussed above, the Group A designation is based on the total weight of the available evidence. The consistency of results that are seen in the numerous studies examined lead to a certainty of greater than 99.9% that secondhand smoke increases the risk of lung cancer in nonsmokers.

Use of what is called in statistics a one-tailed test of significance,"

which often corresponds to a 90% confidence interval, is a standard and appropriate statistical procedure in certain circumstances. The one-tailed test" is used when there is prior evidence that if there is an effect from a substance, it is highly likely to be an adverse rather than a protective effect, or vice versa. In the case of secondhand smoke, an extensive database exists for direct smoking indicating that if chemically similar secondhand smoke also has a lung cancer effect, this effect is likely to be similarly adverse. EPA used one-tailed significance tests for lung cancer in both external drafts of the risk assessment document as well as the final report. Ninety percent confidence intervals were also used in other EPA cancer risk assessments, including methylene chloride, coke oven emissions, radon, nickel, and dioxin.

In the non-cancer respiratory effects portions of the report, two-tailed tests" and 95% confidence intervals were used, since there was less prior evidence from smokers to suggest that secondhand smoke would cause bronchitis, pneumonia, and ear infections in children.

Top of page

The Meta-analysis

Meta-analysis was used for the lung cancer data as an objective method of combining results from many studies and was specifically endorsed by the SAB for use with this database. Some critics argue both that the meta-analysis was not an appropriate technique, and that had EPA included the Brownson study (addressed above) in the meta-analysis of overall spousal exposure, EPA could not possibly have classified secondhand smoke as a known human carcinogen. This just isn't true.

The finding that secondhand smoke is a known cause of lung cancer in humans is based on all the evidence and is not dependent on the meta-analysis of the simple ever- vs. never-exposed comparisons, as the critics suggest. If the meta-analysis were removed from the report entirely, the findings would be precisely the same. The meta-analysis was used primarily for estimating and quantifying the population risks from exposure to secondhand smoke, and an alternative approach also used in the report gave very similar results.

Top of page

Confounders

In the secondhand smoke report, a confounder would be a specific factor that could be responsible for the lung cancer increases observed in nonsmokers instead of secondhand smoke. The tobacco industry and its consultants have suggested, for example, that nonsmoking wives might share in the same poor dietary habits as their smoking husbands, increasing their risk.

The consistency of results across different countries where lifestyle factors, including diet, vary, argues against confounding. For example, while the tobacco industry theorizes that a high fat diet is a confounding factor, the studies from Japan, where dietary fat intake is among the lowest in the world, show a strong dose-response relationship for secondhand smoke and lung cancer.

The EPA report did examine the available data for six potential confounders such as occupation, dietary factors, and history of lung disease, and concluded that none was likely to explain the lung cancer increases seen in the studies.

The 1994 Fontham et al. study controlled for diet and other potential confounders, and concluded, These observations indicate that the strong association in this study between adult secondhand smoke exposure and lung cancer risk cannot be attributed to any likely confounder.

Top of page

"The Threshold Theory"

Although some have argued that tobacco smoke cannot cause cancer below a certain level, there is no evidence that this threshold exists. In the absence of such evidence, carcinogens at any level are considered by EPA to increase risk somewhat, although the degree of risk certainly is reduced as exposure decreases. The increased risks observed in the secondhand smoke epidemiology studies are further evidence that any threshold for secondhand smoke would have to be at very low levels.

Top of page

"Cigarette Equivalents"

The tobacco industry uses the cigarette equivalent" method of comparing smokers' and nonsmokers' exposures to a single component of tobacco smoke to infer that a nonsmoker's exposure to tobacco smoke is insignificant. However, the cigarette equivalent method has no scientific support, and was rejected by the SAB panel that reviewed the EPA report. Among the many problems with this method is the fact that while secondhand smoke and mainstream smoke contain the same approximately 4,000 compounds, their ratios of individual compounds differ by factors in the thousands. Thus, there is no single compound in tobacco smoke that is an adequate indicator for drawing such comparisons. An RJ Reynolds newspaper ad, while utilizing the method, acknowledges it may not be relevant for assessing risk from secondhand smoke.

Top of page

Residential Exposures Translated to the Workplace

The tobacco industry frequently argues that because most studies were based on residential exposures, secondhand smoke has not been shown to be a hazard in the workplace. A substance capable of causing cancer in one environment is certainly capable of causing it in any other environment where exposures are comparable, as is the case with residential and workplace exposure to secondhand smoke. In fact, the 1994 Fontham study found a slightly higher risk for workplace exposure than for residential exposures.

Top of page

The Congressional Research Service (CRS) Report

The RJ Reynolds' media campaign cites a report prepared by the Congressional Research Service (CRS) on cigarette taxes to fund health care reform to argue that CRS believes that the epidemiological evidence on secondhand smoke and health effects is weak and uncertain." However, CRS has not taken a position on either EPA's risk assessment or the health effects of passive smoking.

Two economists from CRS, citing material largely prepared by the tobacco industry, included a discussion of EPA's risk assessment in an economic analysis of a cigarette excise tax proposal to fund health care reform. In EPA's view, the CRS economists' cursory look at the issues is not comparable to the exhaustive analyses and rigorous review process which EPA undertook when examining the extensive database on secondhand smoke and respiratory health. EPA is confident that a comprehensive analysis of the secondhand smoke database by expert scientists from CRS, with adequate peer review, will come to conclusions about the risks of secondhand smoke similar to those of EPA and many other organizations.

Top of page

Cigarette Prohibition

The claim that the government is attempting to bring back prohibition -- this time for cigarettes -- is a complete fabrication and utter nonsense. EPA's interest is to provide information to protect the nonsmoker from involuntary exposure to a hazardous substance. Having a choice to take a risk for themselves should not permit smokers to impose a risk on others.

Top of page

For Further Information

For additional information on secondhand smoke and other indoor air pollutants, contact EPA's:

Indoor Air Quality Information Clearinghouse [IAQ INFO]
at 1-800-438-4318 or
(703) 356-4020 in Washington Metro area
PO Box 37133
Washington DC 20013-7133
(703) 356-5386 (fax)
iaqinfo@aol.com

Top of page

Smoke-free Homes Program

Where You Live A to Z Index

Health Effects

The Smoke-free Homes Pledge

Prometa no fumar en su hogar

Developing Smoke-free Homes Programs

Frequent Questions

Publications and Materials

Links

Contact Us

Frequently Asked Questions

(For more information on the health effects of children's exposure to secondhand smoke, click on Health Effects)

Frequent Questions on Other Indoor Air Topics

Asthma
Indoor Air Quality
Mold/Mold Resources
Radon
IAQ Tools for Schools

Table of Contents

If you have further questions, you can contact Lou Witt in the Indoor Environments Division at (202) 343-9051 or via e-mail at witt.lou@epa.gov

Is there a difference between environmental tobacco smoke and secondhand smoke?

No, both terms mean the same thing – exposure to smoke from the burning end of a tobacco product or exhaled by a smoker. The terms side-stream smoke or passive smoking are also used.

Top of page

How long do the harmful elements in environmental tobacco smoke linger?

Depending on a room's ventilation the smell of tobacco smoke can linger for some time. If tobacco smoke is in the air, it can and does present potential health problems to all, but particularly children. If it is not currently in the air but has been allowed to

permeate clothing, furniture, and furnishing, the odor may still be present and irritating. The volatile and semi-volatile compounds that are unhealthy would not be present in levels high enough to be of concern.

Top of page

How is children's health affected by exposure to environmental tobacco smoke?

EPA's findings, along with a number of other American and international studies, show a negative effect on children's health from environmental tobacco smoke exposure. Environmental tobacco smoke can cause bronchitis and pneumonia, wheezing and coughing spells, more inner ear infections, as well as more frequent and severe asthma attacks. Also, exposure has been associated with sudden infant death syndrome (SIDS).

Top of page

What's EPA's position on children's exposure to environmental tobacco smoke?

EPA's position is that exposure to environmental tobacco smoke increases the health risks for children particularly under the age of six. EPA encourages that until a person can quit, especially parents and caregivers, they should go outside to protect their children. In other words, even if the parents and/or caregivers choose to smoke, they should choose to not smoke (or allow others to smoke) in the home or car. Consequently, EPA's activities are designed to raise awareness among smokers not to smoke around children or allow others to do so. Activities also include encouraging people, smokers and non-smokers, to maintain a smoke-free home and car for their children. Environmental tobacco smoke exposure is particularly harmful to children, causing more inner ear infections, bronchitis, pneumonia, wheezing and coughing spells, and more frequent and severe asthma attacks.

Top of page

What is EPA doing to reduce children's exposure to environmental tobacco smoke?

EPA has authority to conduct a voluntary program to educate the public on the health risks of children's exposure to environmental tobacco smoke. EPA focuses on protecting young children in the home and car because these are the areas they spend the most time in during the ages of 0-6 years.

The messages, "Make your home and car smoke free" and "Until you can quit, go outside for your kids" are key in encouraging parents and caregivers to commit to making and maintaining smoke-free homes and cars. This is the basis for EPA's Smoke-free Homes Pledge Campaign, where people pledge to make their homes and car smoke free. To date, thousands of people have taken the Pledge. People interested in taking the Smoke-free Homes Pledge may do so at either 1-866-SMOKE-FREE (1-866-766-5337) or on the Pledge page

EPA has a wide variety of outreach and educational materials and products that motivate and educate people on protecting children from exposure to environmental tobacco smoke. You can access them by clicking on Publications and Related Links.

Top of page

Can EPA ban smoking or otherwise limit exposure to environmental tobacco smoke?

No. EPA's specific mission is defined by the US Congress. EPA does not have a mandate to regulate smoking or control tobacco in any manner. As it stands now, tobacco control and smoking restrictions are strictly state and local issues. However, based on an Executive Order smoking is prohibited in all federal buildings and facilities.

Top of page

Who has the authority to ban or limit exposure to environmental tobacco smoke?

A number of state and local governments have passed legislation or instituted regulations to ban smoking in work places and public places such as restaurants, bars, and parks. This may include state prisons, local jails, and other detention facilities in some areas. Your local jurisdiction may be the most responsive resource for inquiries. Your state or local health department may be able to provide you with information on smoking prohibition policies and activities in your area. Contact information for specific state indoor air quality or environmental offices can be located at epa.gov/iaq/whereyoulive.html.

Top of page

Why does environmental tobacco smoke come in from my neighbor's apartment?

Read your lease carefully looking for "safe premises" or

"nuisance" clauses or a "covenant of habitability" and get advice from a legal center since smokers in many places don't have a right to smoke in apartments (condominiums are different) and landlords in many cases do have the power to ban smoking. You might also get advice from your local health department since there may be state or county building codes that relate to sanitary and health conditions. Your local chapter of the American Lung Association (1-800-LUNG-USA) also may be able to provide you with additional information on smoking codes and/or ordinances in your area.

Environmental tobacco smoke can come into your apartment in multiple ways. In general, air leakage (i.e. air leaking from outside the walls of a structure, technically known as infiltration) rates for individual apartments depend upon height above the ground, orientation, wind speed, and indoor-outdoor temperature differences, imbalance between supply and exhaust air, stack effects, and differences in ventilation rates between upper and lower floors. What this means is that many activities (fragrant cooking, applying perfume or cologne, smoking, etc.) occurring in one apartment can be shared with other apartments for many different reasons.

Top of page

- About Us
- Publications Catalog
- Surgeon General's Reports
- Research, Data, and Reports
- How To Quit
- Educational Materials
- New Citations
- Tobacco Control Program Guidelines & Data
- Celebrities Against Smoking
- Sports Initiatives
- Campaigns & Events
- Smoking and Health Database
- Related Links

Secondhand Smoke

Esta página en Español

Fact sheet

February 2004

Definition

- Secondhand smoke, also known as environmental tobacco smoke (ETS), is a mixture of the smoke given off by the burning end of tobacco products (sidestream smoke) and the smoke exhaled by smokers (mainstream smoke).[1,2]
- Secondhand smoke contains a complex mixture of more than 4,000 chemicals, more than 50 of which are known or probable human cancer-causing agents (carcinogens).[1,2]
- People are exposed to secondhand smoke in the home, workplace, and in public venues such as bars, bowling alleys, and restaurants.[3]

Health Effects

- Secondhand smoke is associated with an increased risk for lung cancer and coronary heart disease in nonsmoking adults.[1,2,4] Secondhand smoke is a known human carcinogen (cancer-causing agent).[2,4]
- Because their lungs are not fully developed, young children are particularly susceptible to secondhand smoke. Exposure to secondhand smoke is associated with an increased risk for sudden infant death syndrome (SIDS), asthma, bronchitis, and pneumonia in young children.[1,5]

Current Estimates

- An estimated 3,000 lung cancer deaths and more than 35,000 coronary heart disease deaths occur annually among adult nonsmokers in the United States as a result of exposure to secondhand smoke.[6]
- Each year, secondhand smoke is associated with an estimated 8,000–26,000 new asthma cases in children.[4] Annually an estimated

150,000–300,000 new cases of bronchitis and pneumonia in children aged less than 18 months (7,500–15,000 of which will require hospitalization) are associated with secondhand smoke exposure in the United States.[4]

- Approximately 60% of non-smokers in the United States have biological evidence of secondhand smoke exposure.[7]
- Among children aged less than 18 years, an estimated 22% are exposed to secondhand smoke in their homes, with estimates ranging from 11.7% in Utah to 34.2% in Kentucky.[8]

References

1 National Cancer Institute. *Health Effects of Exposure to Environment Tobacco Smoke.* Smoking and Tobacco Control Monograph No. 10 (PDF - 71k). Bethesda, MD: U.S. Department of Health and Human Services, National Institutes of Health, National Cancer Institute; 1999. NIH Pub. No. 99-4645. Accessed: February 2004.

2 National Toxicology Program. *10th Report on Carcinogens.* Research Triangle Park, NC: U.S. Department of Health and Human Services, Public Health Service, National Toxicology Program, December 2002. Accessed: February 2004.

3 Pirkle JL, Flegal KM, Bernert JT, Brody DJ, Etzel RA, Maurer KR. Exposure of the U.S. population to environmental tobacco smoke: The Third National Health and Nutrition Examination Survey, 1988 to 1991. *Journal of the American Medical Association* 1996;275 (16):1233–1240.

4 U.S. Environmental Protection Agency. *Respiratory Health Effects of Passive Smoking: Lung Cancer and Other Disorders.* Washington, DC: U.S. Environmental Protection Agency;1992. Pub. No. EPA/600/6-90/006F. Accessed: February 2004.

5 U.S. Department of Health and Human Services. *Women and Smoking: A Report of the Surgeon General.* Rockville, MD: U.S. Department of Health and Human Services, Public Health Service, Office of the Surgeon General; 2001. Accessed: February 2004.

6 CDC. Annual smoking-attributable mortality, years of potential life lost, and economic costs—United States, 1995–1999 (PDF - 225k). *Morbidity and Mortality Weekly Report* 2002;51(14):300–303. Accessed: February 2004.

7 CDC. *Second National Report on Human Exposure to Environmental Chemicals: Tobacco Smoke.* Atlanta, GA: U.S. Department of Health and Human Services, CDC, National Center for Environmental Health; 2003:80. NCEH Pub No. 03-0022. Accessed: February 2004.

8 CDC. State-specific prevalence of cigarette smoking among adults, and children's and adolescents' exposure to environmental tobacco

smoke—United States, 1996 (PDF – 266k). *Morbidity and Mortality Weekly Report* 1997;46(44):1038–1043. Accessed: February 2004.

Note: More recent information may be available at the CDC'S Office on Smoking and Health Web site: http://www.cdc.gov/tobacco.

For Further Information

Office on Smoking and Health
National Center for Chronic Disease Prevention and Health Promotion
Centers for Disease Control and Prevention
Mailstop K-50
4770 Buford Hwy., N.E.
Atlanta, GA 30341-3717
770-488-5705
http://www.cdc.gov/tobacco

Media Inquiries: Contact the CDC's Office on Smoking and Health press line at 770-488-5493.

One or more documents on this Web page is available in Portable Document Format (PDF). You will need Acrobat Reader (a free application) to view and print these documents.

This page last reviewed August 24, 2005

United States Department of Health and Human Services
Centers for Disease Control and Prevention
National Center for Chronic Disease Prevention and Health Promotion
Office on Smoking and Health

- About Us
- Publications Catalog
- Surgeon General's Reports
- Research, Data, and Reports
- How To Quit
- Educational Materials
- New Citations
- Tobacco Control Program Guidelines & Data
- Celebrities Against Smoking
- Sports Initiatives
- Campaigns & Events
- Smoking and Health Database
- Related Links

Exposure to Environmental Tobacco Smoke and Cotinine Levels — Fact Sheet

- The National Institutes of Health's National Toxicology Program's 9th issue of the Report on Carcinogens listed ETS as a "known" human carcinogen, which indicates that there is a cause and effect relationship between exposure and human cancer incidence.

- ETS is classified as a Group A carcinogen (known to cause cancer in humans) under the EPA's carcinogen assessment guidelines. Exposure to ETS causes lung cancer and has been linked to an increased risk for heart disease in nonsmokers.

- ETS causes about 3,000 lung cancer deaths annually among adult nonsmokers. Scientific studies have also estimated that ETS accounts for as many as 35,000 deaths from ischemic heart disease annually in the United States. More research is needed to know exactly how recent changes in ETS exposure may affect lung cancer rates among adult nonsmokers.

- ETS causes serious respiratory problems in children, such as greater number and severity of asthma attacks and lower respiratory tract infections. ETS exposure increases the risk for sudden infant death syndrome (SIDS) and middle ear infections for children.

- Cotinine is a major metabolite of nicotine. Exposure to nicotine can be measured by analyzing the cotinine levels in the blood, saliva, or urine. Since nicotine is highly specific for tobacco smoke, serum cotinine levels track exposure to tobacco smoke and its toxic constituents.

- In 1991, data showed that nearly 90 percent of the U.S. population had measurable levels of serum cotinine in their blood. The Centers for Disease Control and Prevention's National Report on Human Exposure to Environmental Chemicals found more than a 75 percent decrease in median cotinine (metabolized nicotine) levels for nonsmokers in the United States since 1991.

- Children and teenagers, 3-19 years old, had higher levels of cotinine than did adults, 20 years old and above.

- Involuntary exposure to ETS remains a common, serious public health hazard that is entirely preventable by adopting and enforcing appropriate regulatory policies. Smokefree environments are the most effective method for reducing ETS exposure. Healthy People 2010 objectives address this issue and seek optimal protection of

nonsmokers through policies, regulations, and laws requiring smoke-free environments in all schools, work sites, and public places.

- Based on the way their laws are implemented in practice, California, Connecticut, Delaware, Maine, Massachusetts, New York, Rhode Island, and Washington meet the nation's Healthy People 2010 objective to establish smoke-free indoor air laws covering public places and worksites. Because of comprehensive state laws, virtually all indoor workplaces in these states are now smoke free, including restaurants and bars.

- The dramatic declines in serum cotinine levels among nonsmokers are a good indication that efforts to ensure clean indoor air through smoking restrictions in workplaces, restaurants and other public places are working. However, there are still too many people, especially young people, who continue to be exposed to environmental tobacco smoke (ETS).

This page last reviewed November 28, 2005

United States Department of Health and Human Services
Centers for Disease Control and Prevention
National Center for Chronic Disease Prevention and Health Promotion
Office on Smoking and Health

- About Us
- Publications Catalog
- Surgeon General's Reports
- Research, Data, and Reports
- How To Quit
- Educational Materials
- New Citations
- Tobacco Control Program Guidelines & Data
- Celebrities Against Smoking
- Sports Initiatives
- Campaigns & Events
- Smoking and Health Database
- Related Links

Secondhand Smoke in Your Home

Benefits of a Smoke-Free Home | How to Make (and Keep) Your Home Smoke-Free | Don't Forget Schools and Day Care

We spend more time in our homes than anywhere else. So the thought of cancer-causing chemicals circulating throughout our houses and apartments can be quite unsettling. Yet, according to the Environmental Protection Agency, that is exactly what happens when someone lights a cigarette in your home.

Those most affected by secondhand smoke are children. Because their bodies are still developing, exposure to the poisons in secondhand smoke puts children in danger of severe respiratory diseases and can hinder the growth of their lungs. On top of that, the effects can last a lifetime.

Ventilation systems in homes cannot filter and circulate air well enough to eliminate secondhand smoke. Blowing smoke away from children, going into another room to smoke, or opening a window may help reduce children's exposure but will not protect them from the dangers of secondhand smoke.

Benefits of a Smoke-Free Home

The greatest benefit, of course, is that you will remove all the health risks associated with secondhand tobacco smoke. Plus

- When your home is smoke-free, it will smell much better.
- Your food will taste better.
- You'll spend less time, energy, and money cleaning your curtains, walls, windows, and mirrors.
- Your insurance rates may be lower—check with your insurance company.
- Even your pets will be happier. For example, secondhand smoke increases the risk of lung cancer in dogs.

Return To Top

How to Make (and Keep) Your Home Smoke-Free

- It may feel awkward at first to tell people not to smoke in your home—no one wants to make guests uncomfortable—but if you simply explain the facts about secondhand smoke, they should understand completely. Tell them that for the sake of your family's health, you simply cannot allow smoking in your home. Have gum or mints available as an alternative to lighting up. If visitors absolutely must smoke, tell them they can do so outside.

- If someone in your household smokes, be sympathetic and understanding—but encourage him or her to quit. Let that person know that cigarette smoke affects everyone, not just the smoker. Let them know you care and you want to help. Again, if someone absolutely must smoke, ask that person to do so outside.

Return To Top

Don't Forget Schools and Day Care

- Make sure your child's school and day care programs are smoke-free. And insist that babysitters not smoke around your children.

Return To Top

Related Links

../../ETS_Toolkit/index.htm

This page last reviewed January 26, 2005

United States Department of Health and Human Services
Centers for Disease Control and Prevention
National Center for Chronic Disease Prevention and Health Promotion
Office on Smoking and Health

Home
Public Places
Workplaces

What is Secondhand Smoke?

Secondhand smoke (SHS), sometimes referred to as environmental tobacco smoke (ETS), is a mixture of the smoke given off by the burning ends of a cigarette, pipe, cigar, bidis, and kreteks (sidestream smoke) and the smoke emitted at the mouthpiece and exhaled from the lungs of smokers (mainstream smoke).[1,2]

On This Page

The widespread practice of smoking in buildings exposes nonsmoking occupants to combustion by-products under conditions where airborne contaminant removal is slow and uncertain. Over the past two decades, medical science has shown that nonsmokers suffer many of the diseases of active smoking when they breathe secondhand smoke.

Environmental Tobacco Smoke contains at least 250 chemicals known to be toxic or cause cancer. Unfortunately, the general public's exposure to secondhand smoke is much higher than most people realize.

Return to Top

Working to Reduce Secondhand Smoke Exposure

In January 2000, the U.S. Department of Health and Human Services launched *Healthy People 2010*, a comprehensive, nationwide health promotion and disease prevention agenda. *Healthy People 2010* contains 467 objectives designed to serve as a road map for improving the health

Case 1:04-cv-00879-SJM-SPB Document 63-4 Filed 02/17/2006 Page 26 of 45

of all people in the United States during the first decade of the 21st century.

Several of these objectives relate to tobacco use and exposure to secondhand smoke. One objective is to reduce nonsmoker exposure to secondhand smoke from 65% to 45% nationwide by 2010.

Return to Top

You Can Make a Difference

This toolkit is designed to provide you with the tools you need to take action to reduce secondhand smoke in your community, whether you are

- New to local advocacy.
- An experienced advocate tackling the issue of secondhand smoke for the first time.
- A member of an active coalition already familiar with the issue.

To be most effective, collaborate with a coalition in your area. Contact the local branch of health organizations such as the American Heart Association,* American Cancer Society,* or American Lung Association.* They can let you know about groups you can join in your area, and they may already have begun efforts to tackle the problem of secondhand smoke in your community.

If you find that your community does not have a coalition working on this issue, you can learn how to Build a Coalition* so that your efforts will be effective.

Return to Top

Getting the Information You Need

The Public Places section of this toolkit is complete with

- Action Steps to follow to successfully implement a clean indoor air policy for public buildings in your community—with direct links to the tools you need to complete these steps.
- Tools and materials that you can reproduce for your campaign.
- An extensive Resources section where you can get more information on demand.
- *Best Practices* information to help you make your campaign successful.
- Key reports and other data about Secondhand Smoke.

Check out the Fact Sheet for the most recent statistics and research on secondhand smoke.

The Workplaces section is also complete. It is organized similarly to the Public Places section, except that it also contains information for employers and employees.

Return to Top

More to Come...

Additional information will be added to the toolkit in the months ahead. Be sure to come back and get what you need to tackle the issue of secondhand smoke in

- Restaurants and bars
- Schools

Return to Top

For Information on Secondhand Smoke Exposure in the Home

For information on reducing secondhand smoke exposure in the home, see the information that the U.S. Environmental Protection Agency provides on this topic at http://www.epa.gov/iaq/ets/.

Return to Top

Footnotes

1. U.S. Environmental Protection Agency, Indoor Environments Division. *Indoor Air Quality (IAQ).* Last updated May 29, 2002. Accessed July 24, 2002.

2. U.S. Department of Health and Human Services, National Toxicology Program. *9th Report on Carcinogens, January 2001*. 2001. Accessed July 24, 2002.

Return to Top

Thanks

Special thanks to the *Americans for Nonsmokers' Rights* and the *Minnesota Smoke-Free Coalition*.

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* Links to non-Federal organizations are provided solely as a service to our users. Links do

not constitute an endorsement of any organization by CDC or the Federal Government, and none should be inferred. The CDC is not responsible for the content of the individual organization Web pages found at this link.

Home | Introduction | Action Steps | Tools | Organizations | Best Practices | Links |

Privacy Policy | Accessibility

TIPS Home | What's New | About Us | Fact sheets | Site Map | Contact Us

CDC Home | Search | Health Topics A-Z

This page last reviewed June 17, 2005

United States Department of Health and Human Services
Centers for Disease Control and Prevention
National Center for Chronic Disease Prevention and Health Promotion
Office on Smoking and Health

HOME/OVERVIEW

LUNG CANCER INFORMATION

- Types of Lung Cancer
  - Non Small Cell Lung Cancer
  - Small Cell Lung Cancer
  - Asbestos Lung Cancer
- Lung Cancer Symptoms
- Lung Cancer Staging
- Lung Cancer Treatment Options

MESOTHELIOMA

- Pleural Mesothelioma / Peritonial
- Mesothelioma Symptoms and Diagnosis
- Mesothelioma Staging
- Mesothelioma Clinical Trials

CANCER HOSPITALS

- Locations By State
- Questions and Information From Your Doctor

AT RISK JOBS

CANCER INFORMATION RESOURCES

- Mesothelioma News
- Patient Stories
- Web Resources
- Glossary of Terms

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LUNG CANCER & MESOTHELIOMA

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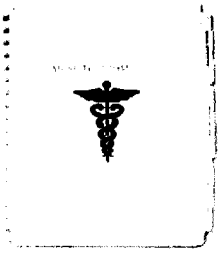
Lung Cancer Symptoms

- A cough that doesn't go away and gets worse over time
- Constant chest pain
- Coughing up blood
- Shortness of breath, wheezing, or hoarseness
- Repeated problems with pneumonia or bronchitis
- Swelling of the neck and face
- Loss of appetite or weight loss
- Fatigue

These lung cancer symptoms may be caused by lung cancer or by other conditions. It is important to check with a doctor.

To help find the cause of lung cancer symptoms, the doctor evaluates a person's medical history, smoking history, exposure to environmental and occupational substances such as asbestos, and family history of cancer. The doctor also performs a physical exam and may order a chest x-ray and other tests. If lung cancer is suspected, sputum cytology (the microscopic examination of cells obtained from a deep-cough sample of mucus in the lungs) is a simple test that may be useful in detecting lung cancer. To confirm the presence of lung cancer, the doctor must examine tissue from the lung. The removal of a small sample of tissue for examination under a microscope by a pathologist can show whether a person has lung cancer. A number of procedures may be used to obtain this tissue:

FREE Mesothelioma Packet



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questions and send a free packet with additional information on:

- New treatment options
- New clinical trials
- Doctors
- Hazardous jobs and products
- Financial Assistance

- Bronchoscopy. The doctor puts a bronchoscope (a thin, lighted tube) into the mouth or nose and down through the windpipe to look into the breathing passages. Through this tube, the doctor can collect cells or small samples of tissue.
- Needle aspiration. A needle is inserted through the chest into the lung cancer tumor to remove a sample of tissue.
- Thoracentesis. Using a needle, the doctor removes a sample of the fluid that surrounds the lungs to check for cancer cells.
- Thoracoscopy. Surgery to open the chest is sometimes needed to diagnose lung cancer. This procedure is a major operation performed in a hospital.

Find out more about...

Lung Cancer Staging

To Obtain the Best Treatment Info & Financial Assistance contact us for a FREE INFORMATION PACKET which includes;

Cancer Hospital Locations
Clinical Trials
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New Treatment Options
Doctors
Financial Assistance

Fill out the form below or call 1-800-780-2686.

Use the "tab" key to move to the next field, not enter.

First Name
Last Name
Address
City
State

Zip

Phone

Email

Have you or a loved one been diagnosed or have:

Mesothelioma: ○ Yes ◉ No

Symptoms of Mesothelioma: Display symptoms ○ Yes ◉ No

Lung Cancer: ○ Yes ◉ No

Fluid in the Lungs/ Pleural Effusion: ○ Yes ◉ No

Did you or your loved one work around asbestos?: ○ Yes ◉ No

Comment / Info Request

Submit and wait...

Please just hit Submit once, then wait for the form to be sent

Site Map | Mesothelioma | Lung Cancer | Non-small cell lung cancer | Small cell lung cancer | Asbestos Lung Cancer | Lung Cancer Symptoms | Mesothelioma News | Pleural Mesothelioma | Mesothelioma Symptoms

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Case 1:04-cv-00379-SJM-SPB Document 65-4 Filed 02/17/2006 Page 31 of 43

Physicians *for a* Smoke-Free Canada

Health Effects of Chemicals found in Cigarette Smoke

Click on a chemical in the table below to view a short summary of its properties and health effects. A source list of studies on which this summary is based can be obtained by contacting or you can download the PDF version by clicking here.

AMMONIA	HYDROGEN CYANIDE
1,3-BUTADIENE	HYDROQUINONE
1-AMINONAPHTHALENE	ISOPRENE
2-AMINONAPHTHALENE	LEAD
3-AMINOBIPHENYL	METHYL ETHYL KETONE
4-AMINOBIPHENYL	NAT
ACETALDEHYDE	NICKEL
ACETONE	NICOTINE
ACROLEIN	NITRIC OXIDE
ACRYLONITRILE	NNK
BENZENE	NNN
BENZO[a]PYRENE	PHENOL
BUTYRALDEHYDE	PROPIONALDEHYDE
CADMIUM	PYRIDINE
CARBON MONOXIDE	QUINOLINE
CATECHOL	RESORCINOL
CHROMIUM	STYRENE
CRESOL	TAR
CROTONALDEHYDE	TOLUENE
FORMALDEHYDE	

AMMONIA

- Ammonia possesses a sharp, irritating odor detectable as low as 1 ppm.\
- At high concentrations, ammonia causes intense irritation, severe eye damage, and asthma.

top

2-AMINONAPHTHALENE

- 2-aminonaphthalene causes cancer in humans.
- There is no safe exposure limit for 2-aminonaphthalene.
- Absorption of 2-aminonaphthalene occurs both by inhalation and through the skin.
- Because it causes cancer, the industrial use of 2-aminonaphthalene is restricted or banned.

top

1-AMINONAPHTHALENE

- 1-aminonaphthalene has been shown to cause lung, liver and leukemia cancers in animals.
- 1-aminonaphthalene may cause cancer in humans.
- 1-aminonaphthalene has been shown to have moderate toxicity in fish.
- Absorption occurs both by inhalation and through the skin.
- Absorption through the skin may occur without a sense of irritation or other warning.
- The main industrial uses of 1-aminonaphthalene include dyes, rubber, and weed control.

top

4-AMINOBIPHENYL

- 4-aminobiphenyl is confirmed to cause cancer in humans.
- The carcinogenic nature of 4-aminobiphenyl has been known since at least 1974.
- This chemical has been called 'one of the most potent known bladder carcinogens'.
- There is no known safe level of 4-aminobiphenyl.
- Absorption occurs through the skin.

- 4-aminobiphenyl is no longer produced on a commercial scale for use in industry.

top

3-AMINOBIPHENYL

- 3-aminobiphenyl is a mutagen, and causes mutation in microorganisms.

top

BENZO[a]PYRENE

- Benzo[a]pyrene (B[a]P) is suspected to cause cancer in humans.
- There is a significant correlation between B[a]P exposure and lung cancer mortality.
- B[a]P was found to cause cancer in animals and fish in every study to date.
- Animal studies showed that as low a dose as 0.05 mg B [a]P caused tumors.
- B[a]P deposits in the lung. Elimination of B[a]P from the lung is severely restricted by cigarette smoking.
- Cancer is more likely to occur with repeated B[a]P exposures than with a single dose of the same amount.
- B[a]P exposure may also cause skin cancer, dermatitis, photoallergy, non-neoplastic respiratory disease and emphysema.
- Exposure to B[a]P results in decreased reproductive capacity for both males and females.
- Absorption of B[a]P also occurs through the skin.

top

FORMALDEHYDE

- Formaldehyde is suspected to cause cancer in humans.
- Formaldehyde occurs naturally at 0.12 to 0.38 parts per BILLION [ppb]. Sidestream smoke increases this by 0.23 to 0.27 parts per MILLION [ppm] (a 1000+ increase).
- Long-term exposure at levels greater than 0.1 ppm appears to be a risk for cancers of the lung, pharynx, buccal cavity, liver, bone, skin, prostate gland, bladder, kidney and eye, leukemia and Hodgkin's disease.
- Animal studies showed formaldehyde is an irritant to eyes, nose, throat and lungs, and causes cellular changes in the upper respiratory tract, a decrease in respiratory rate, and adversely affects the liver.
- Formaldehyde exposure greater than 0.22 ppm is linked to respiratory symptoms such as cough, phlegm, chronic

bronchitis, asthma, shortness of breath and chest colds.

- Formaldehyde is known to produce allergic reactions and induction of asthma-like conditions, lightheadedness, dizziness, diminished dexterity, itching eyes, dry and sore throats, disturbed sleep, unusual thirst, and malignant disease in humans.
- Human eyes are sensitive to formaldehyde at concentrations of 0.01 ppm, and are irritated by formaldehyde at concentrations of 0.05 to 0.5 ppm.
- The main uses of formaldehyde in industry include fertilizer, dyes, disinfectants, germicides, preservatives, and embalming fluid.

top

ACETALDEHYDE

- Studies have shown that acetaldehyde causes cancer in animals, and may cause cancer in humans.
- Small amounts of acetaldehyde irritate the eyes, skin, and respiratory tract of humans and animals.
- Animal studies in which pregnant rats were exposed to acetaldehyde found that acetaldehyde interfered with the exchange of nutrients from the mother to the placenta, resulting in growth retardation, malformation, delayed bone growth and death of the fetus.
- Acetaldehyde may increase the absorption of the other hazardous chemicals in tobacco smoke into the bronchial tubes.
- The main industrial uses of acetaldehyde include silvering of mirrors, leather tanning, fuel, glue, dyes, plastics and synthetic rubbers.
- Acetaldehyde decomposition products include carbon monoxide.

top

ACETONE

- Acetone is an irritant to eyes, nose and throat.
- Acetone irritates, dries, and may burn skin.
- Absorption occurs both via inhalation and through the skin.
- Higher doses can cause dizziness, lightheadedness, damage to the liver and kidneys.

top

ACROLEIN

- Acrolein has not been found to cause cancer. However, in the body, acrolein produces glycidaldehyde which

does cause cancer.

- Long term inhalation studies on animals found that acrolein causes emphysema and inflammation of the lung, liver and kidney.
- Acrolein is intensely irritating to the eyes and upper respiratory tract in human and animals. Acrolein is 5 times stronger an irritant than formaldehyde, acetaldehyde or crotonaldehyde (all of which are found in tobacco smoke).
- The main industrial uses of acrolein include polyurethane manufacture, polyester resins, herbicides and tear gas.

top

PROPIONALDEHYDE

- Inhalation of propionaldehyde causes severe irritation of the respiratory system.
- Propionaldehyde causes irritation to skin and eyes.

top

CROTONALDEHYDE

- Crotonaldehyde is known to cause cancer in animals.
- Crotonaldehyde causes cancer by interfering with DNA function (a genotoxic carcinogen).
- Crotonaldehyde is a fast-acting (within seconds) irritant to the nose and upper respiratory tract.
- The main use of crotonaldehyde in industry is as a warning agent in fuel gases.

top

METHYL ETHYL KETONE

- Methyl ethyl ketone causes nose, throat, and eye irritation in humans at moderate levels.
- The odor of methyl ethyl ketone is detectable at 10 ppm.
- The main uses of methyl ethyl ketone in industry include solvents, resins, artificial leather, rubbers, lacquers, varnishes and glues.

top

BUTYRALDEHYDE

- Butyraldehyde is an irritant to eyes, nose, throat and lungs.
- Higher doses of butyraldehyde causes dizziness and

lightheadedness, and may burn skin.
- The main industrial uses of butyraldehyde include resins, solvents and plasticizers.

top

HYDROGEN CYANIDE

- Hydrogen cyanide causes nasal irritation, confusion, headache, dizziness, weakness and nausea in humans at moderate doses.
- At higher doses, hydrogen cyanide causes asthenia, vertigo, loss of weight and gastrointestinal problems.
- The main uses of hydrogen cyanide in industry include fumigation, as an insecticide, electroplating, metallurgy and photography.

top

NICKEL

- Inhalable, insoluble nickel is confirmed to cause cancer in humans.
- Up to 5% of the general population are sensitized (allergic) to nickel.
- Nickel inhalation increases the risk of cancer or of gastrointestinal symptoms.
- Exposure to inhalable nickel may result in chronic irritation of the upper respiratory tract or bronchial asthma.
- Nickel inhalation exposure increases susceptibility to respiratory infection, allergic contact dermatitis, and pulmonary edema.
- The main uses of nickel in industry include production of stainless steel, alloys, electroplating, coinage, and alkaline batteries.

top

LEAD

- Lead is known to cause cancer in animals.
- Lead may cause cancer in humans.
- Lead is toxic, and soluble in body fluids when inhaled.
- Lead interacts with enzymes, especially those associated with heme synthesis (blood).
- Absorption of low levels of lead causes an increase in blood pressure in humans.
- Lead causes anemia at blood levels above 80 ug/dl.
- Lead poisoning effects on the brain may not be reversible.

- Long term exposure to lead may lead to kidney disease.
- Lead is a possible Reproductive Toxin.
- Lead may affect sperm formation (at greater than 11.9 ug/dl blood lead).
- Lead exposure affects the development of fetuses. Children who were exposed to blood lead levels of greater than 10 ug/dl in the womb have been found to have developmental effects such as depressed intellectual development.
- Air to blood lead levels: 0.03 to 0.19 ug/dl blood per mg/m3 of lead in air.
- The main uses of lead in industry include alloys (solder, bronze, brass), paint pigments, storage batteries, glass, plastics, ceramics.

top

CADMIUM

- Cadmium is confirmed to cause cancer in humans.
- Cadmium primarily targets the kidneys.
- Chronic cadmium exposure is linked to gastrointestinal symptoms, anemia, rhinitis, discoloration of teeth, microfractures, pulmonary emphysema and kidney disease.
- The main industrial uses of cadmium include metal coatings, bearings, reactor control rods, storage batteries, television phosphors, semiconductors, pigments, and dry film lubricants.

top

CHROMIUM

- Cr VI compounds are recognized to cause cancer.
- Cr VI compounds can easily pass into the cell through the cell membrane.
- Cr VI compounds are sensitizers, and can therefore induce an allergic reaction in some individuals.

top

NITRIC OXIDE

- Nitric oxide reacts with haemoglobin to hinder oxygen uptake in the blood.
- Nitric oxide reacts with haemoglobin 1400 times more effectively than carbon monoxide reacts with haemoglobin.
- The toxicity of nitric oxide when combined with carbon monoxide (also in tobacco smoke) is additive.

top

PYRIDINE

- Pyridine vapour causes eye and upper respiratory tract irritation in humans.
- Exposure to pyridine results in an increased production of blood platelets.
- Longer duration exposure to pyridine causes nausea, headache, insomnia, nervousness, and abdominal discomfort in humans.
- The disagreeable odor of pyridine is detectable at less than 1 ppm.
- The main industrial uses of pyridine include solvents, pesticides and resins.

top

QUINOLINE

- Quinoline causes genetic mutations (mutagen) and therefore may increase your risk of cancer.
- Repeated exposure damages the retina of the eye, affecting vision.
- Repeated exposure to quinoline may damage the liver.
- Quinoline exposure may lead to allergy, with rash and itching (sensitizer).
- Quinoline is irritating to the eyes, nose, throat and bronchial tubes, and may cause sore throat, nose bleeds, cough and phlegm.
- Absorption occurs both by inhalation and through the skin.
- Quinoline bioaccumulates in the tissues of fish.
- The main industrial uses of quinoline include dyes, catalysts, insecticides, herbicides, corrosion inhibitors and to preserve anatomical specimens.

top

HYDROQUINONE

- Exposure to hydroquinone leads to eye injury, skin irritation and central nervous system effects in humans.
- The main uses of hydroquinone in industry include rubber production, photography, paints, varnishes and in motor fuel.

top

RESORCINOL

- Resorcinol was found to be irritating to skin and eyes in humans.
- The main industrial uses of resorcinol include tanning, photography, resins, dyes, laminates and adhesives.

top

CATECHOL

- Catechol, when inhaled with benzo[a]pyrene (also found in tobacco smoke), is co-carcinogenic.
- Catechol causes increased blood pressure, upper respiratory tract irritation and eczematous dermatitis in humans.
- At higher doses, catchol causes kidney damage and convulsions.
- The main uses of catchol in industry include photography, rubber, dye, oil, insecticides, and inks.

top

PHENOL

- Studies have shown phenol to be toxic to the respiratory, cardiovascular, hepatic, renal and neurological systems of animals.
- Higher doses of phenol may damage the lungs and central nervous system and induce convulsions in humans.
- Phenol is irritating to the skin, mucous membranes and eyes in humans.
- Phenol may be absorbed by inhalation or through the skin.
- The main industrial uses of phenol include chemicals and drugs, disinfectants, germicidal paints and slimicides.

top

CRESOL

- Cresol was found to promote tumors in mice.
- Cresol is strongly irritating to skin, and causes dermatitis in humans.
- Long term exposure to cresol leads to headaches, nausea, vomiting, elevated blood pressure, impaired kidney function, blood-calcium imbalance and marked tremors, in humans.
- Cresol is absorbed through the skin.
- The main uses of cresol in industry include ore flotation, disinfectants, synthetic resins, dyes, fumigants, and explosives.

top

TAR

Tar is the tobacco industry term for all non-gaseous, non-nicotine, non-water chemicals in tobacco smoke.

top

NICOTINE

- Free-base nicotine (in tobacco smoke) is absorbed almost instantly by inhalation, ingestion and skin contact.
- Nicotine concentrates in the brain, the kidney, the stomach mucosa, the adrenal medulla, the nasal mucosa and the salivary glands.
- Studies show that nicotine exposure can result in seizures, vomiting, depressions of the central nervous system, growth retardation, developmental toxicity in fetuses, and preterm birth with reduced body weight and brain development in animals.
- Nicotine is excreted in breast milk.
- Mild nicotine poisoning in humans results in the following symptoms: vomiting, diarrhea, increase in respiration, heart rate, blood pressure, headache, dizziness, and neurological stimulation.
- Nicotine is considered responsible for many of the acute psychological and physiological effects of smoking, chewing or inhaling tobacco.
- The main uses of nicotine in industry (besides tobacco) include insecticides (now mostly banned) and as tranquilizing darts for wildlife.

top

CARBON MONOXIDE

- Tobacco Smoke is the major source of personal inhalation of carbon monoxide.
- Carbon monoxide is absorbed into the blood, resulting in reduction in exercise tolerance, increased angina and headaches.
- Carbon monoxide binds to haemoglobin, reducing the oxygen-carrying capacity of the blood.
- As little as 3% absorbed carbon monoxide in haemoglobin results in decreased psychomotor function, and therefore can impair driving skills. Headaches may occur at 10% carbon monoxide in haemoglobin.
- Carbon monoxide binds to myoglobin, decreasing heart and muscle function.
- Carbon monoxide is a possible Reproductive Toxin.

- Studies on pregnant animals show decreased birth weights, fetal death or damage at moderate levels of carbon monoxide.
- Fetal carbon monoxide levels are generally 10 to 15% higher than maternal levels.
- Inhaled tobacco smoke increases the level of carbon monoxide in the fetus, increasing the chance of low birth weight, and possible perinatal death or retardation of mental abilities.

top

1, 3-BUTADIENE

- 1,3-butadiene suspected to cause cancer in humans.
- Joint exposure to styrene (also found in tobacco smoke) may increase the risk of disease.
- The toxicity of 1,3-butadiene is increased by prolonged or repeated exposures.
- The main industrial uses of 1,3-butadiene include synthetic rubber and tire manufacture.

top

ISOPRENE

- Isoprene causes skin, eye and mucous membrane irritation.

top

ACRYLONITRILE

- Acrylonitrile is suspected to cause cancer in humans.
- Acrylonitrile is highly toxic. It is similar to cyanide in toxicity, and is also known as 'vinyl cyanide'.
- Absorption of acrylonitrile is from the respiratory and gastrointestinal tract and through the skin.
- Studies on pregnant animals showed 'a significant maternal toxicity', leading to increased possibility of deformation in the fetus and offspring.
- The main industrial uses of acrylonitrile include manufacture of bottles and as a fumigant for tobacco.
- In the United States, acrylonitrile has been withdrawn as a fumigant for all other food commodities.

top

BENZENE

- Benzene is confirmed to cause cancer in humans.
- Benzene is known to cause leukemia in humans.
- Cumulative exposure to benzene is the most likely predictor of the possibility of developing leukemia.
- Leukemia may manifest 2 to 50 years after exposure to benzene.
- Benzene is highly toxic.
- Benzene produces chromosomal aberrations in humans and in animals.
- Benzene is absorbed through the skin.
- Previously, benzene was used in industry to manufacture inks, rubber, lacquers and paint remover.

top

TOLUENE

- Toluene is highly toxic.
- Toluene is a possible Reproductive Toxin.
- Inhaled toluene appears in blood circulation within 10 seconds and accumulates in body fat.
- Toluene is a depressant to the central nervous system in animals and in humans.
- Long term low level exposure results in headaches, lassitude, loss of appetite, disturbances in menstruation, reductions in intelligence and psychomotor skills.
- Higher exposure results in encephalopathy, headache, depression, lassitude, impaired coordination, transient memory loss, impaired reaction time, dizziness, nasal discharge, drowsiness, and metallic taste.
- The main uses of toluene in industry include rubbers, oils, resins, adhesives, inks, detergents, dyes, and explosives.

top

STYRENE

- Styrene is a possible human carcinogen.
- Styrene has been found to produce headaches, ocular and conjunctival irritation and slowed reaction time, fatigue, dizziness and nausea, reduced attention and manual dexterity, and reductions in colour discrimination, in humans.
- Reproductive effects of styrene include a possible increased incidence of spontaneous abortion and increased number of abnormal sperm.
- When styrene and butadiene (also in tobacco smoke) are combined, they produce 4-phylglycolhexene, a suspected sensitizer.
- The main industrial uses of styrene include plastics,

coatings, polyesters, resins, and synthetic rubbers.

top

NNN

- NNN (N-nitrosonornicotine) is a carcinogenic Tobacco-Specific Nitrosamine (TSNA) found only in tobacco products.
- NNN is formed from nicotine directly and is the most abundant cancer-causing TSNA.
- NNN is a yellow, oily liquid that is known to cause nose, throat, lung and digestive tract cancer in animals.
- NNN may cause reproductive damage in humans.-These is no safe level of exposure to NNN.

top

NNK

- NNK [(4-methylnitrosamino)-1-(3-pyridyl)-1-butanone] is a carcinogenic Tobacco-Specific Nitrosamine (TSNA) found only in tobacco products.
- NNK is a powerful lung carcinogen.
- NNK induces adenoma and AC tumors of the lung.
- There is no safe level of exposure to NAT.

top

NAT

- NAT (N-nitrosoanatabine) is a possibly carcinogenic Tobacco-Specific Nitrosamine (TSNA) found only in tobacco products.

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